IJSPT

ORIGINAL RESEARCH

DEFORMATION RESPONSE OF THE ILIOTIBIAL BAND-TENSOR FASCIA LATA COMPLEX TO CLINICAL-GRADE LONGITUDINAL TENSION LOADING *IN-VITRO*

Mark Wilhelm, PT, DPT[1]
Omer Matthijs, PT, ScD[2]
Kevin Browne, PT, ScD[1]
Gesine Seeber, PT, MS[3]
Anja Matthijs, PT, MS[2]
Phillip S. Sizer, PT, PhD[1]
Jean-Michel Brismée, PT, ScD[1]
C. Roger James, PhD[1]
Kerry K. Gilbert, PT, ScD[1]

ABSTRACT

Background: Iliotibial Band (ITB) syndrome is a troublesome condition with prevalence as high as 12% in runners. Stretching has been utilized as a conservative treatment. However, there is limited evidence supporting ITB elongation in response to a stretching force.

Purpose/Hypotheses: The purpose of this study was to describe the iliotibial band tensor fascia lata complex (ITBTFLC) tissue elongation response to a simulated clinical stretch *in-vitro*. The authors hypothesized that the ITBTFLC would undergo statistically significant elongation when exposed to a clinical-grade stretching regimen, with the majority of the elongation occurring within the proximal ITBTFLC region.

Study Design: Within subjects repeated measures *in-vitro* design.

Methods: The strain response of six un-embalmed ITBTFLCs to a simulated clinical stretch of 2.75% elongation was assessed. Four sets of array marks were placed along the length of the ITBTFLC. Photographic images were taken in resting position (with 1.0% *in-situ* elongation) and with an additional 2.75% elongation. Tissue elongation was compared between proximal, middle, and distal ITBTFLC regions.

Results: A paired samples *t-test* demonstrated a significantly longer ITBTFLC in the "stretched" versus resting condition ($p = 0.001$). Significant elongation was observed in the proximal (3.96mm (SD = 1.35); $p = 0.001$), middle (2.12mm (SD = 1.49); $p = 0.018$) and distal (2.25mm (SD = 1.37); $p = 0.01$) regions during the "stretched" versus the resting condition. A one-way ANOVA demonstrated a significant main effect for region ($p = 0.002$). The proximal region exhibited significantly greater elongation versus the middle ($p = 0.003$) and distal ($p = 0.007$) regions, with no significant difference between the middle and distal regions ($p = 0.932$).

Conclusion: The results of this study demonstrate that the ITBTFLC is capable of elongation in response to a clinically simulated stretch. The proximal ITB region underwent significantly greater elongation than the middle and distal regions and may be more likely to respond to "stretching" in clinical situations. Future investigation should assess the ITBTFLC load/deformation properties to determine whether a short-term clinically available stretch translates into permanent tissue elongation.

Key Words: Iliotibial band, iliotibial band syndrome, stretch, tensor fascia lata

Level of Evidence: III

CORRESPONDING AUTHOR
Kerry K. Gilbert, PT, ScD
Professor/Program Director
Doctor of Physical Therapy Program,
Department of Rehabilitation Sciences
School of Health Professions
Texas Tech University Health Sciences Center
3601 4th Street, 6280
Lubbock, Texas, USA
806-743-4525
E-mail: Kerry.gilbert@ttuhsc.edu

[1] Department of Rehabilitation Sciences, Center for Rehabilitation Research, School of Health Professions, Texas Tech University Health Sciences Center, Lubbock, Texas, USA
[2] Boma, Physical Therapy Outpatient Clinic, Mariazeller Straße 30, 8605 Kapfenberg, Styria, Austria
[3] University Hospital for Orthopaedics and Trauma Surgery Pius-Hospital, Medical Campus Carl von Ossietzky University Oldenburg, Oldenburg, Germany

INTRODUCTION

Iliotibial Band Syndrome (ITBS) is a commonly identified clinical condition characterized by focal lateral knee pain, which is often experienced while running and cycling. The prevalence of ITBS is reported to be 12% in runners[1] and comprises 15% of cycling overuse injuries,[2] often resulting in pain levels sufficient to result in activity cessation. Despite the high prevalence and impact on activity participation, controversy persists regarding ITBS etiology, related causal mechanisms, and treatment approaches.

Iliotibial Band Syndrome is thought to originate from a variety of functional anatomical and activity related factors.[1] Anatomical factors may include specific anatomical features and differences such as femoral neck angle, femoral torsion, and Q-Angle. Activity related factors such as overuse and activity load are thought to contribute to ITBS development and pain persistence.[1] The iliotibial band (ITB) is a distal continuation of the fascia arising from the tensor fascia lata (TFL), gluteus maximus, and gluteus medius muscles.[3] The ITB serves as a supportive fascial structure that proximally encapsulates the TFL muscle and spans from the iliac crest and gluteus maximus to Gerdy's (anterolateral tibial) tubercle. The ITB is considered to be a lateral thickening of the circumferential fascia lata.[4] The ITB longitudinal fibers are continuous with the fascial sheath completely enveloping the lateral thigh and adhere to the entire length of the lateral intermuscular septum that attaches onto the femur's linea aspera, separating the anterior and posterior compartments of the thigh.[5] The ITB is linked to the femur through obliquely oriented strands of dense, regular fibrous connective tissue.[5] After coursing between the biceps femoris and vastus lateralis the ITB attaches to the lateral femoral condyle and sends lateral retinacular fibers to the patella.[6] Deep to the ITB at the lateral femoral condyle, investigators describe the presence of a bursa,[7] retro-fascial space,[8] and a fat pad that is highly vascularized, containing Pacinian corpuscles and nerve fibers.[3,5,9] Finally, the most distal fibers attach to the Gerdy's (anterolateral tibial) tubercle.

The histologic structure of the ITB is consistent with tendinous tissue,[9] where the amount of elastic fibers within the tendon is sparse.[5] Despite diligent review of literature, no comprehensive report of the tensile properties of the ITBTFL complex was found. However, a 1931 study examined cadaveric fascia lata tissue and reported that 8-10% elongation would lead to a structural "break".[10] Gratz likened the tremendous tensile strength of the fascia lata to "soft steel wire of similar weight", yet with "unexpected degrees of elasticity".[10] Falvey et al[4] showed that hip motion (hip flexion, knee flexion, and hip adduction) produced the most ITB strain, compared to straight leg raise or Ober's test position, however, tensile properties of anatomically different regions of the ITBTFLC in total were not assessed.

Changes in the structural characteristics and mechanical properties of the ITB have been described as substantial contributors to development and persistence of ITBS.[2,11-13] Selected authors purport tissue dysfunction and aberrant biomechanics as causative factors in ITBS development.[14-16] Orchard et al[17] described ITBS as an impingement between the lateral femoral condyle and the ITB posterior margin at approximately 30 degrees of knee flexion just prior to heel strike while running. The impingement event was thought to occur as the distal ITB repetitively moved in an anterior-posterior direction over the lateral femoral condyle, producing friction.[1,17,18] Conversely, Fairclough and colleagues[19] challenged the notion of an anterior-posterior movement, and instead proposed that the ITB moves in a lateral to medial direction causing lateral knee compressive forces.[5] These authors reported that a relative tensioning and relaxation of the anterior and posterior fibers occurs during knee flexion and extension, contributing to tissue microtrauma and subsequent symptoms.

Management strategies have been suggested for reducing ITB dysfunction and improving mechanics, with limited evidence as to their efficacy.[1] Clinical stretching techniques have been discussed as a possible treatment component aimed at modifying tissue dysfunction. However, limited evidence has been published supporting the effect of stretching on mechanical changes and or symptoms.[12,20-22] Doucette and Goble[23] reported a correlation between ITBS symptom improvement and increased ITB flexibility as measured by the distance from the medial patella to the table during Ober's position. Conversely, other investigators have suggested that ITB stretching is not beneficial for reducing symptoms on a long-term

basis.[24] Noehren et al[25] suggested that increased closed chain hip adduction and internal rotation moments contribute to ITB syndrome and propose hip abductor strengthening as a management strategy. Grau et al[26] observed decreased hip adduction in ITBS subjects while running and proposed hip abductor stretches as a treatment approach. Yet, none of these studies examined the actual mechanical effects of stretching on the ITB tissue.

To clarify the ITB stretching effect, Willet et al[27] discovered that the mid-thigh ITB does not appear to be the exclusive constraint to hip adduction during the Ober's test, with the gluteus medius, gluteus minimus, and the joint capsule constraining adduction. While these investigators examined ITB behaviors that constrained hip adduction, no study has examined the elongation potential of the entire ITB-tensor fascia lata complex (ITBTFLC) during simulated clinical stretching elongation. Therefore, the purposes of this study were to: (1) determine if the *in-vitro* un-embalmed ITBTFLC elongates in response to clinical-grade longitudinal tension loading and (2) describe the elongation behaviors found in different tissue regions along its ITBTFLC length. The authors hypothesized that the ITBTFLC would undergo elongation when exposed to clinical-grade stretching loads, and that the majority of the elongation would occur at the proximal portion, which includes the TFL muscular fibers.

MATERIALS AND METHODS

Preparatory Procedures

Six right-sided ITBTFLCs were harvested from six un-embalmed human cadavers (3 male and 3 female) with a mean age of 81.5 (SD ± 7.8) ranging from 73 to 92 years. Cadaver characteristics including cause of death can be found in Table 1. All cadavers were stored at 3.9˚C prior to ITBTFLC removal. In order to establish the resting tension present in the intact cadaveric ITBTFLC, pre- and post-dissection length measures were conducted both with and without intermuscular septum dissection. On the cadaver's left lower extremity, the entire ITBTFLC was exposed and the resting length of the ITBTFLC was subsequently determined. The resting length was determined in the following manner: the dissecting investigator separated the ITBTFLC from the underlying structures by cutting the intermuscular septum. The investigators then located the most distal (inferior and posterior) prominence of Gerdy's tubercle and the most proximal insertion of the ITBTFLC into the iliac crest (posterior fibers). A pin was placed at each location and ITBTFLC length was measured using a tape measure to record the *in-situ* resting length of the ITBTFLC. Investigators then removed Gerdy's tubercle (with the pin in place) and the length between pins was measured again. The second investigator held the tubercle and the ITBTFLC in place with sufficient tension to remove slack while avoiding a detectable stretch. The average of three measures was recorded both before and after tubercle dissection. Following these measurements, the ITBTFLC was removed along with a small portion of the iliac crest.

The difference in ITBTFLC length between the pre- and post- Gerdy's tubercle dissection was divided by the original length (before dissecting Gerdy's tubercle from the tibia) and multiplied by 100% to calculate the ITBTFLC percent change in length after removing Gerdy's tubercle from the tibia under each of the two dissection methods. The following formula was used for this calculation:

$$\%\Delta\text{Length} = \frac{\text{Length}_{(\text{Original})} - \text{Length}_{(\text{Gerdy's cut})}}{\text{Length}_{(\text{Original})}} \times 100$$

Table 1. *Cadaver characteristics.*

Subject Number	Age	Sex	Cause of Death and Comorbidities
1	89	Female	Complications of vascular dementia, COPD
2	92	Male	COPD, CAD, CHF, CAV
3	76	Female	COPD, CAD, CHF, West Nile Virus, Post-Polio Syndrome, Adult Failure to Thrive
4	83	Male	CVA, HTN
5	73	Female	Pancreatic Cancer
6	76	Male	Failure to Thrive, Dementia, Crohn's Disease, prostate Cancer

COPD = Chronic Obstructive Pulmonary Disease, CAD = Coronary Artery Disease, CHF = Congestive Heart Failure, CAV = Cardiac Allograft Vasculopathy, CVA = Cerebrovascular Accident, HTN = Hypertension

This calculation represents ITBTFLC *in-situ* elongation and was used to "pre-tension"/mimic the *in-situ* length of ITBTFLC prior to the simulated "clinical stretching" protocol.

The *in-situ* "clinical stretch" elongation percent was calculated in a similar manner to the *in-situ* resting length (described above) by measuring changes in ITBTFLC length from resting position to full hip adduction in the frontal plane. Pilot testing involved one intact cadaveric specimen with the ITBTFLC exposed from the iliac crest to the distal insertion at Gerdy's tubercle. The cadaver was positioned supine with the non-testing lower extremity in hip and knee flexion. Resting length of the ITBTFLC was then measured. The non-testing lower extremity was then flexed at the hip and knee and positioned over the testing leg with the foot positioned lateral to the testing side leg. One investigator stabilized the pelvis while a second investigator adducted the testing lower extremity with maximum force with force placement just distal to Gerdy's tubercle. A third investigator re-measured the ITBTFLC length with maximal adduction in this position resulting in a 2.75% increase in ITBTFLC length compared to resting. This testing position was most similar to "Stretch A" as described by Fredericson and colleagues (2002).[21] Based on the relatively benign nature of this stretching position and its similarity to actual clinical stretches, it is unlikely that this 2.75% elongation would produce injury.

Two custom-made clamping devices were fabricated using a commercially available two-part resin material with an abrasive surface secured to each side of the clamping devices to prevent specimen slippage. Once harvested, ITBTFLC specimens were stored at -18°C until 12 hours prior to data collection when they were transferred to a different storage device and held at 3.9°C. Four hours prior to data collection, each specimen was allowed to warm to room temperature (19.8°C). All specimens were carefully cleaned, removing adipose tissue and muscle fibers that could have interfered with visualization of the ITBTFLC or altered the mechanical properties of the tissue. Specimens were then lightly cleaned with a 70% isopropyl alcohol solution using a soft cloth. Specimens were excluded if observable abnormalities or damage was observed after they were cleaned. Specimens were placed flat on a hard surface and 1.0mm diameter markers were arranged and drawn in a 4-marker array positioned on the lateral surface of the specimen at the following sites (Figure 1): (1) Marker Array 1- distal marker positioned just distal to the musculotendinous junction between TFL and ITB; (2) Marker Array 2- distal marker positioned 250mm distal to Marker Array 1; (3) Marker Array 3- distal marker positioned proximal to marker 1, 50% of the distance from marker 1 to the most proxi-

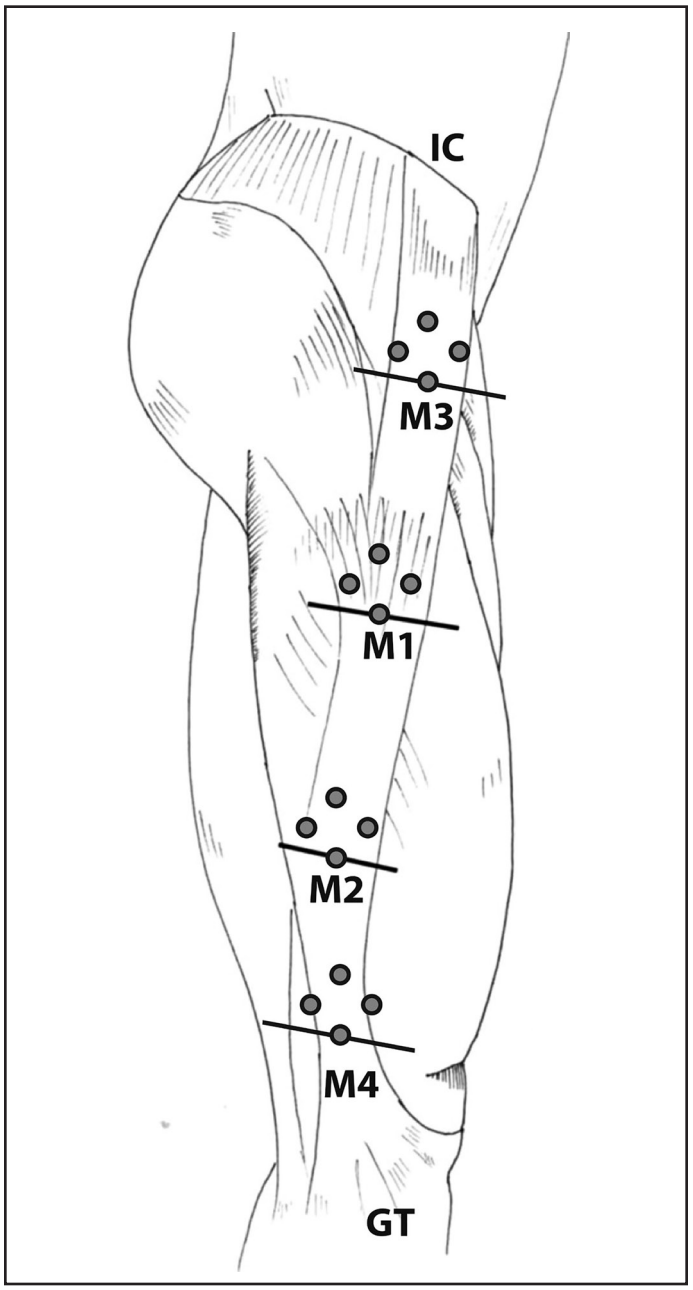

Figure 1. *Placement of Marker Arrays in cadaveric ITBTFLC. M1 = Marker Array 1; M2 = Marker Array 2; M3 = Marker Array 3; M4 = Marker Array 4; IC = Iliac Crest; GT = Gerdy's Tubercle*

mal end of the TFL insertion at the iliac crest; and (4) Marker Array 4- distal marker positioned distal to marker 2, 50% of the distance from marker 2 to the most distal end of the ITB insertion at Gerdy's Tubercle. Each of the three regions were anatomically unique, with the TFL present in the proximal region, the middle region being attached to the linea aspera via the intermuscular septum, and the distal region absent of muscle fibers and having less attachment to the linea aspera versus the middle region. Approximately 50% of the TFL resided between Marker Array 1 and Marker Array 3 with the other 50% falling proximal to Marker Array 3. Additionally, while the ITB in this area serves as an attachment to the gluteus maximus, all gluteus maximus fibers were removed prior to testing.

Investigators mounted each ITBTFLC specimen into a 10 kN material testing system (MTS; MTS Systems Corporation, Eden Prairie, MN, USA) using custom-made clamping devices. The ITBTFLC was mounted with the distal end in the upper bracket and the bony ends of the ITBTFLC just outside the clamping devices to further prevent material slippage (Figure 2). A 12.2-megapixel digital camera (Canon Rebel xsi eos 450D, 18-55mm zoom lens; Canon U.S.A., Inc) was used to capture images. The camera was placed approximately one meter from the plane of the ITBTFLC, with the focal point centered at the specimen's midpoint.

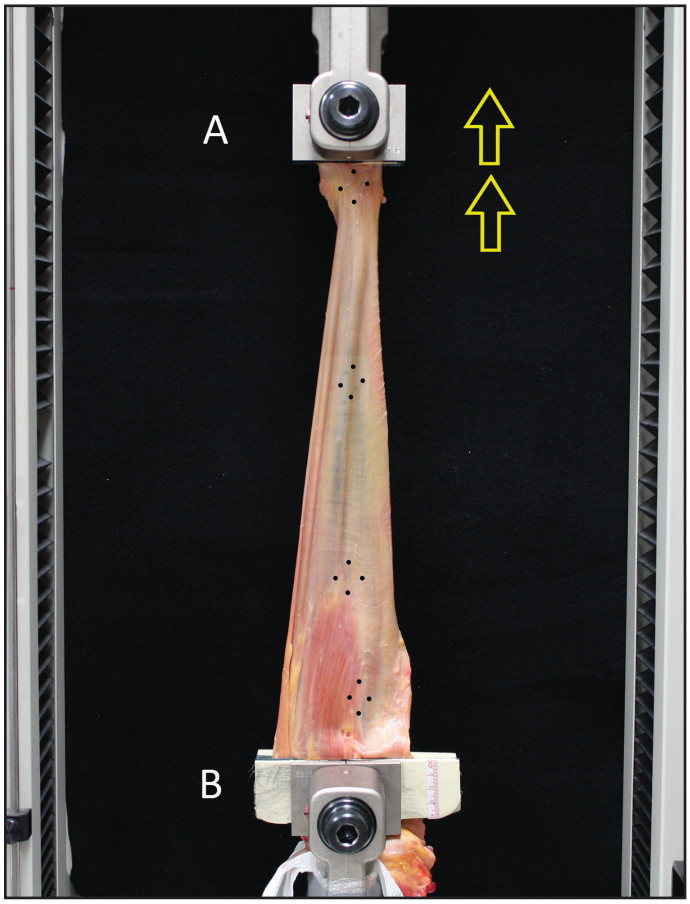

Figure 2. *Mounting the ITBTFLC in the Materials Testing System. "A" represents the distal ITBTFLC end and "B" represents the proximal end. Arrows indicate the direction of mechanical lines of force produced by the MTS.*

Each specimen was mounted in the MTS to a predetermined (1%) *in-situ* elongation and an image was captured. This 1% strain was established through the calculation of the mean ITBTFLC length difference between intact Gerdy's tubercle and cut Gerdy's tubercle during specimen harvesting as described above. Once mounted in the MTS and pre-tensioned, the full length of the specimen was recorded. Next, the specimen was elongated at five mm/second to the simulated "clinical stretch" that equaled an additional 2.75% strain as determined by *in-situ* cadaveric pilot testing described above. This "clinical stretch" position was beyond the pre-tensioned 1% *in-situ* state and maintained by the MTS device for 40 seconds. Afterward, the tissue was returned to the *in-situ* resting length for an additional 40 seconds. The simulated stretch protocol was repeated an additional three times. During each stretch protocol, the length was maintained for 40 seconds to mimic a clinical stretch scenario. A second image was captured at the end of the fourth stretch cycle with the specimen still in the loaded position. After the image was captured, the investigator returned the ITBTFLC to the *in-situ* resting length. Images were imported into and analyzed using a custom MATLAB (The MathWorks, Inc, Natick, MA, USA) program designed to calculate: 1) overall ITBTFLC elongation; and 2) elongation for each ITBTFLC section. Marker locations were identified by the investigator in both images (pre-and post-stretch cycle) and region lengths were calculated with consideration to both vertical and horizontal change using marker coordinates. Elongation was then calculated by comparing pre- to post-stretch cycle region lengths.

Statistical Methods

All data were analyzed using IBM SPSS version 21.0 for Windows (IBM Corp. Armonk, NY). Descriptive

statistics were calculated to summarize the demographic characteristics of the sample. Skewness, kurtosis and the Shapiro Wilk test were used to establish data normality. A paired samples *t-test* was used to determine if a difference existed between the simulated *in-situ* length and the length after the simulated stretch protocol. A one-way ANOVA was performed to identify significant main effects for the ITB region. Tukey's post-hoc pair-wise comparison was used to identify the locations of significant differences. Significance was set at $\alpha = 0.05$ for all statistical comparisons.

RESULTS

All variables were normally distributed as defined by meeting at least two of three criteria (Shapiro Wilk test p-value >0.05, skewness between -2 and +2 and kurtosis between -2 and +2). The ITBTFLC was significantly elongated ($t=-6.753$; $p=0.001$) in the "stretched" condition (381.62 ± 39.58 mm, 95% CI 349.95 – 413.28) versus resting condition (373.03 ± 39.45 mm, 95% CI 341.46 - 404.60), representing an average elongation of 2.3% between the most proximal (M3) and distal (M4) regions. The one-way ANOVA demonstrated a significant main effect for ITBTFLC region [$F(2,15)=9.589$; $p=0.002$]. The post-hoc pairwise comparison tests (Figure 3) demonstrated that the proximal region strain (4.45 ± 1.79% 95%, CI 3.01 - 5.88; includes the TFL) was significantly greater than the middle (1.42 ± 1.02%, 95% CI 0.60 - 2.24; $p=0.003$) or distal (1.70 ± 1.00% 95% CI 0.9-2.49; $p=0.007$) strain values, while there was no significant difference between the middle and distal strain values ($p=0.932$). Average force required to produce 2.75% initial elongation of the initial specimen was 79.15 N ± 41.05. After the initial 40-second simulated stretch cycle, the average force was 61.81 N ± 34.13. Visual specimen inspection did not reveal obvious tissue damage resulting from the 2.75% elongation protocol.

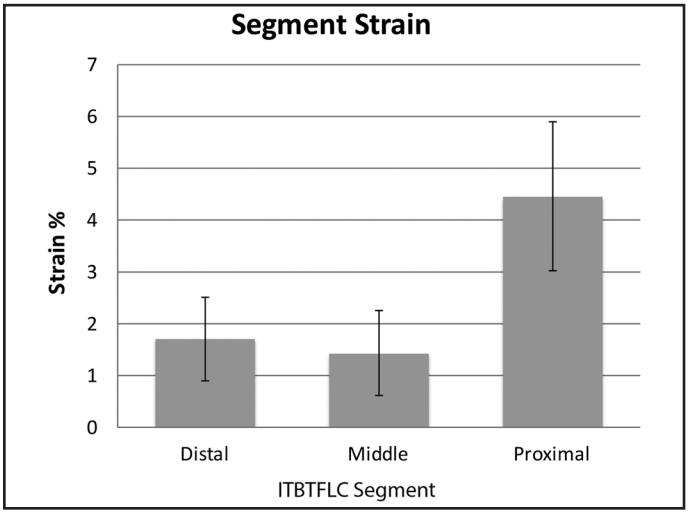

Figure 3. *Strain of each ITBTFLC segment with 95% confidence intervals.*

DISCUSSION

Results of this *in-vitro* study demonstrate that the ITBTFLC complex is capable of undergoing elongation (mean: 2.3%, range:1.1-3.5%) during a clinical-grade longitudinal tension loading protocol, and that significantly greater elongation was found in the proximal region when compared to the middle and distal regions. Initial testing during ITBTFLC specimen harvesting demonstrated a 1% resting elongation *in-situ* and an additional 2.75% elongation during an *in-situ* simulated clinical stretch compared to *in-vitro* length. These same elongation values were used during the "simulated stretch" protocol. Although the outcomes of this study demonstrated greater elongation than those presented in the study by Falvey et al,[4] which demonstrated a 0.23 ± 0.18% mean elongation during a maximum voluntary hip abductor contraction, both studies demonstrate minimal elongation in the ITB itself. While the proximal region (including the TFL) lengthened by 4.45% in the current study, the middle and distal regions elongated by only 1.42% and 1.7%, respectively. The presence of the TFL in the proximal region was likely a factor contributing to the greater elongation observed in this region in the current study, where the location of greatest deformation occurred at the apparent pathway of least resistance, localized in the TFL and its connection with the ITB. This may correspond with the transition from muscle tissue to dense, regular fibrous connective tissue in the specific area.[5] Within the range of loads tested in the present study, the outcomes suggest that most elongation will occur in this (proximal) region versus other regions of persistent higher stiffness. While not statistically significant, the distal region lengthened slightly more than the middle region. Since the intermuscular septum was not present in the current study, speculation regarding the influence of

this ITBTFLC attachment on the elongation pattern *in-vivo* or *in-situ* is not possible and should be considered for further study.

Many different treatment options other than stretching have been suggested for use to increase ITB extensibility. These include, but are not limited to, osteopathic manipulative treatment techniques that include a counterstain technique,[28] self-administered myofascial release techniques utilizing a foam roll, [22,29] and myofascial release techniques that are manually applied by a therapist.[22] However, any contemporary recommendation supporting the use of these approaches can only be based on clinical experience and speculative conclusions in the absence of rigorous clinical data. Future research should examine the mechanical, neurophysiological, and clinical impact of such strategies.

Stretching is a common intervention utilized to affect elongation of the ITB. Little consensus exists regarding the structural impact of ITB stretching on the actual tissues. Different investigators[30,31] have suggested that ITB fibers can be stretched by observing decreased ITB width during a modified Ober's maneuver. While both studies found that *in-vivo* ITB width narrowed during the test, they did not examine actual ITB longitudinal elongation response. Other investigators have examined the effects of stretching on ITB longitudinal deformation in cadaveric specimens. Matsumoto et al.[32] used cadaveric selective cutting to discover that ITB superficial fibers at the mid-thigh level create a notable constraint to ITB elongation under a stretching load. Using mid-thigh ITB strain-gauge measures in unembalmed cadavers, Falvey et al.[4] observed less than five percent ITB deformation occurred during the Ober test and hip flexion-adduction-external rotation and questioned whether stretching produces appreciable clinical stretch or strain and, moreover, whether or not it is capable of producing a lasting ITB lengthening effect. However, these investigators focused their evaluation on the middle portion of the ITB, not the entire ITB length.

While ITBTFLC "stretching" has been shown to be clinically beneficial,[21,23] based on the paucity of comprehensive ITBTFLC tensile strength data, it is difficult to determine where clinical stretching protocols fall relative to the load/deformation curve for this specific tissue. Clinical stretching protocols may produce tissue elongation that falls within the elastic range of tissue deformation, leading to temporary clinical benefits, however, more research is needed to definitively resolve this question. Additionally, it is unlikely that short term "clinical stretching" produces permanent ITB deformation and as a result, other factors such as neuromuscular control changes may contribute to perceived clinical benefits.[4] Future *in-vivo* research should examine the effects of a long-term stretching program on ITB stiffness, thus allowing for longer termed effects such as collagen deposition and cellular level processes involved in tissue healing and regeneration.

Although the current study's findings are in agreement with those of Falvey et al[4] demonstrating minimal stretching of the ITB, these findings are in contrast to those of Frederickson et al,[21] who suggested that three different ITB stretch positions resulted in considerable ITB lengthening. It is important to note that these authors measured lengthening using 3-D motion analysis *in-vivo*, which may have resulted in marker movement due to skin movement in relation to underlying bony landmarks. While these authors reported *in-vivo* ITB lengthening of 9.84-11.15% using surface markers, direct ITBTFLC elongation measurements using a simulated clinical stretch during limited pilot testing in preparation for the current study resulted in only 2.75% elongation. However, the pilot testing stretch procedure was similar to stretch "A" by Frederickson et al,[21] which resulted in 9.84% elongation in their study. Based on analysis of data from the current study as well as the study by Gratz,[10] it is likely that a deformation of 9.84-11.15% could result in clinically significant ITB tissue damage.

There are a few limitations to this study. Tissue properties may be slightly different for *in-vivo* versus *in-vitro* specimens; however, it is difficult to accurately assess the direct length of all three portions of the ITBTFLC *in-vivo*. While the tissue used for the present analysis was harvested from a specimen sample that was older than those individuals who would likely develop ITB pain, it is difficult to obtain cadaveric tissue samples from a younger population for testing. The inclusion of the TFL tissue in the current study limits the ability to apply the findings

specifically to ITB tissue tensile properties in the absence of TFL elongation. Future studies should examine the ITB in the absence of the TFL to better understand the isolated ITB tensile properties. Moreover, future research should examine the influence of tensile properties in tissues surrounding the ITBTFLC. Finally, the cadaveric nature of this study does not allow for assessment of muscular tone influence on ITBTFLC elongation. Future studies should develop means to examine changes in stiffness and deformation within the ITBTFLC *in-vivo*. Future studies should also examine the load deformation behavior of the ITBTFLC and the influence of the intramuscular septum on ITBTFLC behavior.

CONCLUSION

The results of the current study suggest that the ITBTFLC is capable of tissue elongation under normal physiologic loads that simulate a clinical stretching protocol. It is uncertain whether this "stretch" translates into sustained, clinically meaningful tissue elongation. Greater lengthening occurred in the proximal region of the ITBTFLC, suggesting that the proximal region (containing the TFL) is more likely to undergo elongation in response to a clinical stretch force when compared to the middle or distal regions. The increased lengthening response in the proximal region may be due to the presence of the TFL. Results of this study do not challenge the perceived clinical benefit of ITB stretching, but suggest that benefits may be related to changes at the level of the TFL as opposed to the ITB proper.

REFERENCES

1. Ellis R, Hing W, Reid D. Iliotibial band friction syndrome--a systematic review. *Man Ther*. 2007;12(3):200-208.
2. Holmes JC, Pruitt AL, Whalen NJ. Iliotibial band syndrome in cyclists. *Am J Sports Med*. 1993;21(3):419-424.
3. Muhle C, Ahn JM, Yeh L, et al. Iliotibial band friction syndrome: MR imaging findings in 16 patients and MR arthrographic study of six cadaveric knees. *Radiology*. 1999;212(1):103-110.
4. Falvey EC, Clark RA, Franklyn-Miller A, Bryant AL, Briggs C, McCrory PR. Iliotibial band syndrome: An examination of the evidence behind a number of treatment options. *Scand J Med Sci Sports*. 2010;20(4):580-587.
5. Fairclough J, Hayashi K, Toumi H, et al. The functional anatomy of the iliotibial band during flexion and extension of the knee: Implications for understanding iliotibial band syndrome. *J Anat*. 2006;208(3):309-316.
6. Stecco A, Antonio S, Gilliar W, et al. The anatomical and functional relation between gluteus maximus and fascia lata. *J Bodyw Mov Ther*. 2013;17(4):512-517.
7. Ekman EF, Pope T, Martin DF. Magnetic resonance imaging of iliotibial band syndrome. *Am J Sports Med*. 1994;22(6):851-854.
8. Nemeth WC, Sanders BL. The lateral synovial recess of the knee: anatomy and role in chronic Iliotibial band friction syndrome. *Arthroscopy*. 1996;12(5):574-580.
9. Baker RL, Souza RB, Fredericson M. Iliotibial Band Syndrome: Soft Tissue and Biomechanical Factors in Evaluation and Treatment. *PM R*. 2011;3(6):550-561.
10. Gratz CM. Tensile strength and elasticity tests on human fascia lata. *J Bone Joint Surg Am*. 1931;13(2):334-340.
11. Cooperman JM. Case studies: Isolated strain of the tensor fasciae latae. *J Orthop Sports Phys Ther*. 1984;5(4):201-203.
12. Barber DFA, Sutker AN. Iliotibial Band Syndrome. *Sports Med*. 1992;14(2):144-148.
13. Khaund R, Flynn SH. Iliotibial band syndrome: a common source of knee pain. *Am Fam Physician*. 2005;71(8):1545-1550.
14. Louw M, Deary C. The biomechanical variables involved in the aetiology of iliotibial band syndrome in distance runners - A systematic review of the literature. *Phys Ther Sport*. August 2013:1-12.
15. Fredericson M, Cookingham CL, Chaudhari AM, Dowdell BC, Oestreicher N, Sahrmann SA. Hip abductor weakness in distance runners with iliotibial band syndrome. *Clin J Sport Med*. 2000;10(3):169-175.
16. Strauss EJ, Kim S, Calcei JG, Park D. Iliotibial band syndrome: evaluation and management. *J Am Acad Orthop Surg*. 2011;19(12):728-736.
17. Orchard JW, Fricker PA, Abud AT. Biomechanics of iliotibial band friction syndrome in runners. *Am J Sports Med*. 1996;24(3):375-379.
18. Jelsing EJ, Finnoff JT, Cheville AL, Levy BA, Smith J. Sonographic evaluation of the iliotibial band at the lateral femoral epicondyle: Does the iliotibial band move? *J Ultrasound Med*. 2013;32(7):1199-1206.
19. Fairclough J, Hayashi K, Toumi H, et al. Is iliotibial band syndrome really a friction syndrome? *J Sci Med Sport*. 2007;10(2):74-76.
20. Lucas CA. Iliotibial band friction syndrome as exhibited in athletes. *J Athl Train*. 1992;27(3):250-252.

21. Fredericson M, White JJ, MacMahon JM. Quantitative analysis of the relative effectiveness of 3 iliotibial band stretches. *Arch Phys Med Rehab*. 2002;83:589-592.
22. Fredericson M, Weir A. Practical management of iliotibial band friction syndrome in runners. *Clin J Sport Med*. 2006;16(3):261-268.
23. Douciette SA, Goble EM. The effect of exercise on patellar tracking in lateral patellar compression syndrome. *Am J Sports Med*. 1992;20(4):434-440.
24. Geraci MC, Brown W. Evidence-based treatment of hip and pelvic injuries in runners. *Phys Med Rehabil Clin N Am*. 2005;16(3):711-747.
25. Noehren B, Davis I, Hamill J. ASB Clinical Biomechanics Award Winner 2006. *Clin Biomech*. 2007;22(9):951-956.
26. Grau S, Krauss I, Maiwald C, Axmann D, Horstmann T, Best R. Kinematic classification of iliotibial band syndrome in runners. *Scand J Med Sci Sports*. 2011;21(2):184-189.
27. Willett GM, Keim SA, Shostrom VK, Lomneth CS. An Anatomic Investigation of the Ober Test. *Am J Sports Med*. 2016;44(3):696-701.
28. Pedowitz RN. Use of osteopathic manipulative treatment for iliotibial band friction syndrome. *J Am Osteopath Assoc*. 2005;105(12):563-567.
29. Okamoto T, Masuhara M, Ikuta K. Acute effects of self-myofascial release using a foam roller on arterial function. *J Strength Cond Res*. 2014;28(1):69-73.
30. Wang T-G, Jan M-H, Lin K-H, Wang H-K. Assessment of stretching of the iliotibial tract with ober and modified ober tests: An ultrasonographic study. *Arch Phys Med Rehab*. 2006;87(10):1407-1411.
31. Wang H-K, Ting-Fang Shih T, Lin K-H, Wang T-G. Real-time morphologic changes of the iliotibial band during therapeutic stretching; An ultrasonographic study. *Man Ther*. 2008;13(4):334-340.
32. Matsumoto H, Seedhom B. Tension characteristics of the iliotibial tract and role of its superficial layer. *Clin Orthop Relat Res*. 1995;(313):253-255.